Amendments to the Claims

This listing of claims will replace all prior versions, and listings, of claims in the application.

Listing of Claims

1. (Twice Amended) A transgenic [rodent] mouse whose cells contain a DNA sequence comprising:

   (a) a nerve tissue specific promoter operatively linked to a DNA sequence which encodes amyloid-beta peptide binding alcohol dehydrogenase (ABAD), and

   (b) a nerve tissue specific promoter operatively linked to a DNA sequence encoding each of mutant human amyloid precursor proteins hAPP695, hAPP751, and hAPP770 bearing mutations linked to familial Alzheimer's disease in humans,

   wherein said [rodent] mouse exhibits at least one phenotype from the group consisting of: reduced basal synaptic transmission; inhibited synaptic plasticity; increased neuronal stress; elevated 4-hydroxynonenal in cerebral cortex; increased heme oxygenase type I in cerebral cortex; decreased microtubule-associated protein 2 in cerebral cortex; and increased levels of activated caspase 3 antigen in cortical neurons.

2. (Twice Amended) The transgenic [rodent] mouse of claim 1, wherein the promoter of both element (a) and (b) is platelet derived growth factor (PDGF)-B-chain promoter.

3. (Canceled)

4. (Canceled)

5. (Twice Amended) A method for evaluating in a transgenic [rodent] mouse the potential therapeutic effect of an agent for treating Alzheimer's disease in a human, which comprises:

(a) providing an agent to a transgenic [rodent] mouse whose cells comprise

(i) a nerve tissue specific promoter operatively linked to a DNA sequence which encodes amyloid-beta peptide binding alcohol dehydrogenase (ABAD), and

(ii) a nerve tissue specific promoter operatively linked to a DNA sequence encoding each of mutant human amyloid precursor proteins hAPP695, hAPP751 and hAPP 770 bearing mutations linked to familial Alzheimer's disease,

(b) determining the therapeutic effect of the agent on the transgenic [rodent] mouse by monitoring basal synaptic

transmission or synaptic plasticity, wherein an increase in basal synaptic transmission or synaptic plasticity indicates that the agent would have a potential therapeutic effect on Alzheimer's disease in humans.

6. (Original) The method of claim 5, wherein the promoter of both element (a) and (b) is platelet derived growth factor (PDGF)-b-promoter.

7. (Canceled)

8. (Canceled)